

Advancements in Ventilation During Cardiac Resuscitation

Ian R. Drennan, CCP, PhD^{a,b,c,d,e,*}, Matthew R. Neth, MD^f,
Brian K. Walsh, PhD, RRT, RRT-NPS, RRT-ACCS, RPFT, FAARC^g

KEYWORDS

• Ventilation • Cardiac arrest • Resuscitation

KEY POINTS

- Effective ventilation during cardiac arrest is crucial for oxygen delivery and gas exchange, but both hyperventilation and hypoventilation can cause significant harm, including lung injury and neurologic impairment.
- Current guidelines recommend specific ventilation volumes and rates, but there is limited high-quality evidence to define optimal parameters, and practices often deviate from recommendations.
- Challenges in research include the chaotic out-of-hospital environment, measurement difficulties, and patient heterogeneity, which hinder understanding of ideal ventilation strategies.
- Emerging technologies like real-time ventilation feedback devices and capnography show promise in improving ventilation quality, but their impact on patient outcomes remains to be established.
- Future directions should focus on personalized ventilation approaches, better understanding of ventilation parameters, and addressing knowledge gaps through advanced monitoring and targeted research.

^a Interdepartmental Division of Critical Care, Department of Medicine, University of Toronto, Toronto, ON, Canada; ^b Division of Emergency Medicine, Department of Family and Community Medicine, Institute of Health Policy, Management and Evaluation, Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada; ^c Department of Emergency Services, Sunnybrook Research Institute, Sunnybrook Health Sciences Centre, Toronto, ON, Canada; ^d Ornge Critical Care Transport, Mississauga, ON, Canada; ^e FIRST60, Li Ka Shing Knowledge Institute, St Michael's Hospital, Toronto, Canada; ^f School of Medicine, Oregon Health and Science University, Portland, OR, USA; ^g Department of Respiratory Therapy, University of Texas Medical Branch, Galveston, TX, USA

* Corresponding author. Department of Emergency Services, Sunnybrook Research Institute, Sunnybrook Health Sciences Centre.

E-mail address: ian.drennan@sunnybrook.ca

Crit Care Clin ■ (2026) ■-■

<https://doi.org/10.1016/j.ccc.2025.08.007>

criticalcare.theclinics.com

0749-0704/26/© 2025 Elsevier Inc. All rights are reserved, including those for text and data mining, AI training, and similar technologies.



SHOUT
SHARE **IT**

Abbreviations

AOI	airway opening index
ARDS	acute respiratory distress syndrome
CO ₂	carbon dioxide
CPR	cardiopulmonary resuscitation
ETCO ₂	end-tidal carbon dioxide
ILCOR	International Liaison Committee on Resuscitation
MV	mechanical ventilation
OHCA	out-of-hospital cardiac arrest
PBW	predicted body weight
PEEP	positive end-expiratory pressure
RCT	randomized clinical trial
ROSC	return of spontaneous circulation
RR	respiratory rate
Vt	tidal volume

BACKGROUND***Importance of Ventilation and Oxygenation in Cardiac Arrest***

Cardiac arrest remains a leading cause of mortality worldwide, emphasizing the critical need for effective resuscitation techniques. Recent advancements in ventilation technology have the potential to play a pivotal role in improving patient outcomes from cardiac arrest. Understanding the nuances of ventilation while striking a balance between providing adequate support and avoiding complications is essential yet remains elusive.

During cardiac arrest, effective ventilation is important for maintaining oxygen delivery to vital organs, particularly the heart and brain. Proper positive pressure ventilation supports gas exchange, helping to restore normal physiologic function after return of spontaneous circulation (ROSC). The primary goals of ventilation during resuscitation are to ensure adequate oxygenation and facilitate the removal of carbon dioxide (CO₂) without inducing additional injury. There are known consequences of improper ventilation in this vulnerable patient population.

Hyperventilation

Hyperventilation, delivery of large tidal volume (Vt) and/or faster than normal respiratory rate (RR), has a number of potential detrimental effects. High minute ventilation leads to hypocapnia and alkalosis shifting the oxygen-hemoglobin dissociation curve to the left and decreasing oxygen delivery. Excessive stretch, regional lung hyperinflation, and repetitive airway opening play a role in lung injury and place patients at risk for acute respiratory distress syndrome (ARDS).¹ This event, no matter the phase of resuscitation, kicks off a cascade of inflammatory cytokines and other biologic mediators along with hyperoxia that contribute to progressive pulmonary dysfunction, multiple organ failure, and death.^{2,3}

During cardiopulmonary resuscitation (CPR), an increase in intrathoracic pressure created by excessive positive pressure ventilation (ie, hyperventilation) can lead to a decrease in venous return and cardiac output,^{4,5} potentially impacting patient survival. In addition, hyperventilation can mechanically increase pulmonary vascular resistance and injure the lung from excessive pressure (barotrauma), excessive volumes (volutrauma), or poor compliance owing to air trapping from inadequate expiratory times, especially during chest compressions that intermittently obstruct the airways during the passive expiratory phase.

Following ROSC, persistent hyperventilation can lead to subsequent hypocapnia and resultant cerebral vasoconstriction. High intracranial pressure threatens the

integrity of the brain and can adversely affect neurologic outcome. Hyperventilation is used to decrease intracranial pressure by reducing cerebral blood flow and volume. Hyperventilation in patients with traumatic brain injury can result in a 34% decrease in cerebral blood flow and 9% decrease in cerebral blood volume.⁶

Hypocapnia induced by hyperventilation increases cerebral neuronal excitability and seizure duration, increasing oxygenation and glucose consumption, excitatory amino acid production, and anaerobic metabolism.^{7,8} Hyperventilation creates a reduction in oxygen supply and delivery, followed by an increase in oxygen consumption that leads to demand outstripping supply and further brain tissue hypoxia (Fig. 1).

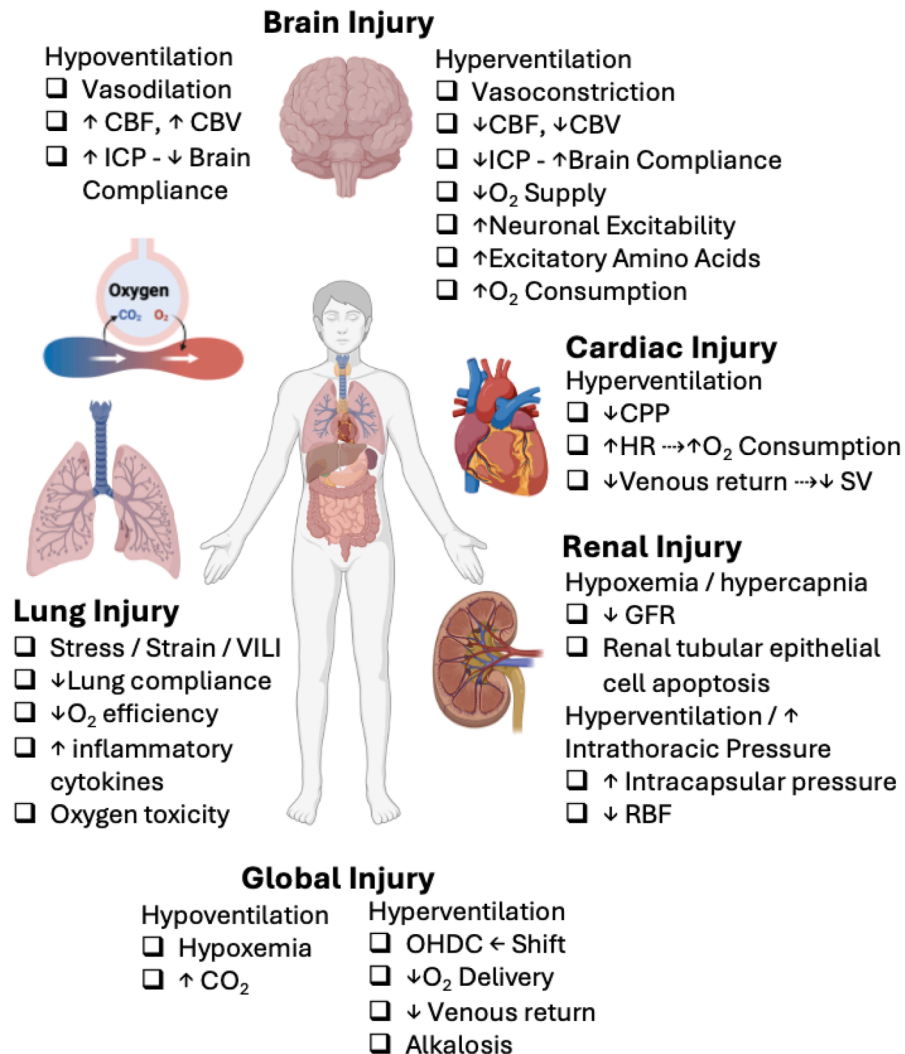


Fig. 1. Effects of abnormal ventilation. CBF, cerebral blood flow; CBV, cerebral blood volume; CPP, cardiac perfusion pressure; GFR, glomerular filtration rate; HR, heart rate; IP, intracranial pressure; RBF, renal blood flow; SV, stroke volume; VILI, ventilator induced lung injury. (Created in BioRender. Walsh, B. (2025) <https://BioRender.com/pkcfvnp>.)

Passive oxygenation and hypoventilation

Chest compressions during CPR are primarily aimed at maintaining circulatory flow but are ineffective as a standalone ventilatory mechanism. During compressions, the lungs collapse because of increased intrathoracic pressure, and they expand upon chest wall recoil, creating a passive ventilation effect. However, this mechanism generates insufficient V_ts to support adequate gas exchange. Studies have shown that compression-only CPR produces V_ts that are far below the requirements for effective alveolar ventilation, reported to be less than 20 mL in adult patients.^{9,10}

Although it has been reported that passive oxygenation may provide short-term benefits in witnessed shockable arrests, this benefit was not observed in unwitnessed arrests or arrests caused by nonshockable rhythms.¹¹ These reported advantages of passive oxygenation in witnessed shockable arrests are likely due to uninterrupted chest compressions rather than the absence of ventilation. These findings underscore the need for active ventilation strategies tailored to optimize gas exchange and patient outcomes during CPR and immediately following ROSC.

Despite the potential negative impact of hypoventilation and hyperventilation on lung injury, gas exchange, and hemodynamics in low flow states, best practice for ventilation in cardiac arrest is controversial. Blood flow during CPR is estimated to be 25% to 33% of normal,¹² yet the ventilation requirements to maintain tissue oxygenation are not known. Targeting normal ventilation parameters (rate and volume) may not only be unnecessary but may predispose patients to further injury from hyperoxia and high airway pressures. Current evidence suggests that providers often ventilate at faster rates than are recommended by guidelines, but that these faster rates are not necessarily associated with worse hemodynamics or patient outcomes.^{13,14} A recent randomized clinical trial (RCT) by Prause and colleagues¹³ challenged the current understanding of ventilation dynamics during cardiac arrest, demonstrating that RRs of 20/min compared with 10/min resulted in higher minute ventilation, but only a small increase in mean airway pressure and no difference in rates of ROSC, median pH, or median P_{CO₂}. This study adds to a body of preclinical research that has questioned the understanding of heart-lung interactions during cardiac arrest and the impact of hyperventilation on intrathoracic pressure and hemodynamics.^{15,16} More research is necessary to evaluate the role of ventilatory rate, volume, and minute ventilation to optimize patient outcomes from cardiac arrest.

Current Evidence for Ventilation During Cardiac Arrest

There is a lack of evidence informing the current understanding of ventilation requirements during cardiac arrest. The American Heart Association guidelines recommend for adult patients in cardiac arrest a volume of ventilation sufficient to cause chest rise, or approximately 500 to 600 mL, and a ratio of 30 chest compressions to two ventilations without an advanced airway, or one ventilation every 6 seconds asynchronously during continuous chest compressions with an advanced airway in place.¹⁷ The European Resuscitation Council similarly suggests providing enough volume to see visible chest rise without specifying an estimated volume, while attempting to avoid gastric insufflation.¹⁸ These recommendations are supported by the International Liaison Committee on Resuscitation (ILCOR) Consensus on Science with Treatment Recommendations.^{19,20} There are no data, however, that suggest these are the optimal ventilation parameters during cardiac arrest.

Research has focused on multiple aspects of ventilation during cardiac arrest, including chest compression to ventilation ratio, the role of passive oxygenation, ventilation rate and volume, and differences in ventilation between basic and advanced airway devices. The quality of evidence varies for the different aspects of ventilation.

Chest compression to ventilation ratios and asynchronous versus synchronous ventilations remain controversial areas in resuscitation. Over time, resuscitation science has moved to increase the number of chest compressions, reducing the number of ventilations that are administered.

A large, randomized trial found no difference in patient survival between continuous chest compressions and 30:2 compressions to ventilations.²¹ Ventilation rate is supported by multiple observational studies.^{14,22} The challenge with all these studies, however, is that none of them were able to measure the actual ventilations delivered. Much of the evidence supporting recommendations for ventilation volume is extrapolated from other areas of medicine, such as anesthesiology and critical care. There is a paucity of research specific to cardiac arrest population. Previous guidelines supported higher ventilation targets, suggesting volumes of 800 to 1200 mL to provide appropriate oxygenation and ventilation during CPR.²³ Subsequent research identified that similar partial pressure of oxygen and partial pressure of carbon dioxide could be obtained with smaller volumes, with less risk of gastric insufflation.²⁴ Guideline organizations therefore started to recommend smaller volumes of ventilation (500–600 mL). There is no evidence, however, that these volumes are associated with improved patient outcomes.

CURRENT CHALLENGES

Research Challenges with Respect to Ventilation

Current research studies examining ventilation during cardiac arrest suffer from several limitations that make interpretation challenging. Most studies are retrospective in nature and suffer from inherent limitations in study design, including significant risk of confounding. These limitations make it difficult to determine whether patient outcomes are due to differences in ventilation, or some other related factor. In addition, cardiac arrest research, which largely occurs in the out-of-hospital setting, is a challenging setting to conduct research. The uncontrolled and chaotic nature of the out-of-hospital environment adds to the complexity of performing high-quality research to examine the importance of ventilation during cardiac arrest.²⁵

Research examining ventilation is highly prone to measurement error, as tools to accurately measure ventilation delivery during manual bag-mask ventilations are limited. It is therefore likely that the ventilations are often not provided as intended. Idris and colleagues²⁶ found that, during basic life support CPR, 30:2 chest compression to ventilation, when EMS providers paused chest compressions to deliver ventilations, during most pauses, less than two ventilations were actually delivered.

Ventilation research is further confounded by the fact that cardiac arrest is a heterogeneous disease in which patients likely have different ventilation requirements based on the cause of the cardiac arrest (cardiac vs respiratory), patient demographics (age, sex), and underlying comorbidities (pulmonary disease). These challenges have contributed to the limited understanding of proper ventilation during cardiac arrest, and there remains considerable knowledge gaps with respect to optimizing ventilation delivery.

Clinical Challenges to Performing High-Quality Ventilation

Manual ventilation is a complex and difficult skill to master. Despite best efforts, high-quality manual ventilation is often not achieved in the out-of-hospital or in-hospital setting. This is especially true with bag-mask ventilation, which has the added complexity of requiring a good mask seal to the patient's face to allow ventilation to occur.

There are several tools and techniques available that can help to ensure effective manual bag-mask ventilation. Oropharyngeal and nasopharyngeal airways can help to alleviate the tongue from the back of the oropharynx and direct air to the trachea. The use of manometers to monitor pressure during ventilation can help to ensure that manual breaths are not delivered with pressures that can lead to alveolar damage or increase the risk of gastric insufflation. Utilization of a two-person bag-mask technique has been shown to be superior compared with single-person ventilation. With two-person bag-mask ventilation, one provider performs the mask seal with the thenar eminences, while the second provider delivers the ventilations in contrast to the standardly taught one-person “E-C clamp” technique. Multiple studies have demonstrated superior ventilations with this two-person technique,^{27–29} especially with inexperienced providers³⁰ and those who have a difficult time obtaining an adequate mask seal (eg, smaller hands or difficult patient anatomy).²⁹

Utilization of pediatric or small adult bag-mask ventilation has been suggested to limit the volume of ventilation and avoid hyperventilation. A recent study by Snyder and colleagues,³¹ however, found that the use of a small adult ventilation bag resulted in lower rates of ROSC than with standard-size adult bag-mask. The effects of minute ventilation, acid base status, and hemodynamics remain unclear and need additional research.³¹

One of the challenges with manual ventilation is that monitoring the quality of ventilations is limited. Beyond visualization of chest rise, there are few tools available to allow for more objective determination of appropriate ventilation. Chest rise on average can be detected with 186 mL of volume and is deemed sufficient at approximately 350 to 400 mL.³² This is a subjective measurement, dependent on the observer, and varies person to person and with the environment in which ventilation is delivered (eg, floor, moving vehicle, stretcher, and so forth). In addition, it can be difficult to administer adequate volume and to visualize chest rise without pausing chest compressions to deliver ventilations; however, current recommendations are to perform continuous chest compressions after an advanced airway has been placed. Therefore, other methods to monitor ventilation quality are needed.

End-tidal carbon dioxide (ETCO₂) monitoring provides valuable information for several aspects of cardiac arrest management but is limited in its ability to guide ventilations. ETCO₂ measurements rely on a complex interaction between metabolism, perfusion, and ventilation. During cardiac arrest, ETCO₂ values reflect cardiac output and deranged metabolism, making it difficult to reliably use ETCO₂ values to guide ventilations. ETCO₂ can, however, be used to count RR as a valuable component of ventilation delivery.

Last, there is a large body of research comparing basic and advanced airway management for delivery of ventilations during cardiac arrest. Overall, the evidence supports either an advanced airway or bag-mask ventilation, as long as the technique used is appropriate for the situation and the provider has adequate training and experience to ensure a high rate of success with limited interruptions in chest compressions.^{33–35} One limitation of all the randomized trials on airway management is that the quality of ventilations was not measured. This remains a major knowledge gap, and presumably at least as important a question as which device providers should use to deliver those ventilations. Despite the neutral trial findings, there are some nuanced reasons advanced airways may be advantageous that were not evaluated in the clinical trials. First, advanced airways allow for continuous chest compressions, which can improve chest compression fraction, a significant predictor of patient outcomes. In addition, advanced airways provide a continuous seal to help limit insufflation of the stomach, while maintaining a patent airway that will allow for better

sampling and lead to more accurate monitoring of ET_{CO_2} . One of the limitations of advanced airways is that it is difficult to monitor chest rise, currently the recommended indicator of sufficient ventilation. As most resuscitations in Advanced Life Support environments receive an advanced airway at some point during the resuscitation, it is imperative to identify additional means of monitoring ventilation quality.

Utility of Capnography and End-Tidal Carbon Dioxide

ET_{CO_2} measurement provides valuable information and has several indications in the management of cardiac arrest. ET_{CO_2} remains the gold standard for confirmation of placement of an advanced airway. In addition, ET_{CO_2} provides valuable information about chest compression quality, the occurrence of ROSC, and prognostication of patient outcomes.

Chest compression quality, most often chest compression depth, has been associated with ET_{CO_2} values with deeper chest compressions providing increased ET_{CO_2} values.^{36,37} ET_{CO_2} values less than 20 mm Hg are often suggested to indicate that chest compressions should be improved during resuscitation.^{38,39}

ET_{CO_2} values that remain less than 10 mm Hg after 20 minutes of advanced life support resuscitation are associated with poor outcomes.⁴⁰ The specificity of this finding, however, does not consistently demonstrate high enough specificity (>99% specificity) to be considered as the sole criteria to terminate resuscitation. Current guidelines recommend using ET_{CO_2} as part of a multimodal approach to determining futility.¹⁷ Other studies have examined the prognostic indication of different aspects of ET_{CO_2} , such as the initial value,^{40,41} the last value, the trend over time,⁴² or the time spent under or over a specific threshold value. None of these ET_{CO_2} values have proven superior in terms of patient prognostication, and the optimal use of ET_{CO_2} is not known.⁴³ As research evolves, it is clear that there is much that is not understood with respect to the complexity of ET_{CO_2} and cardiac arrest management.

Despite the valuable information offered from ET_{CO_2} monitoring during cardiac arrest, the utility of ET_{CO_2} in monitoring or guiding ventilation quality is not known. Previous research has demonstrated that ventilation rate can impact ET_{CO_2} measurement with faster rates resulting in lower ET_{CO_2} readings.^{44,45} However, ET_{CO_2} measurements are significantly confounded by chest compression quality (perfusion), duration of cardiac arrest,⁴² medication administration (epinephrine and sodium bicarbonate),^{46–48} and underlying patient factors,⁴⁹ making it unreliable to use to guide ventilation during cardiac arrest management.

NOVEL CONCEPTS

Ventilation Feedback Devices

Real-time ventilation feedback devices have recently been developed that may help improve the understanding of the optimal ventilation strategy in cardiac arrest. Currently available devices use pressure-based flow sensors to measure and provide calculated values for different ventilatory parameters, such as inspiratory and expiratory V_t , ventilation rate, and air leak, while providing real-time feedback during resuscitation. Utilization of these devices may provide information for advancement in both clinical practice, through the delivery of real-time feedback, and in future research, as a method of evaluating ventilation delivery during clinical trials. A growing number of studies suggest that real-time ventilation feedback can increase compliance with guideline recommendations for ventilation rate and V_t in simulated settings.

Gould and colleagues⁵⁰ performed a before-and-after simulation study using teams of emergency medical technicians. Teams performed two 8-minute rounds of CPR,

first without real-time ventilation feedback followed by a round with real-time feedback. The investigators found that ventilation rate and volume compliance with targets were significantly improved (41% vs 71%; $P < .01$) and (31 vs 79%; $P < .01$), respectively, with the use of real-time visual feedback.⁵⁰ Similarly, subsequent simulation studies have continued to demonstrate improved compliance with ventilation parameter targets with the use of real-time feedback devices.^{51–56} A recent ILCOR scoping review article identified 13 simulation studies and a single before-and-after clinical study examining the use of real-time ventilation feedback in cardiac arrest management.⁵⁷ The findings were consistent across all studies that use of real-time feedback improved ventilation parameters. However, there was a noticeable lack of clinical studies and patient-centered outcomes; thus, no recommendations were made.⁵⁷

Drennan and colleagues⁵⁸ conducted a before-and-after study of the implementation of real-time ventilation feedback in the field in 412 cases of cardiac arrest. They found improved compliance with predefined targets for both ventilation volume and rate with the use of real-time feedback. The investigators, however, did not find any statistical relationship between ventilation parameters and ROSC, although the study was likely underpowered to detect a significant finding.

Although the results of early studies show promise, suggesting improved ventilation with real-time feedback, additional research is necessary to determine the utility of feedback devices in clinical practice. Future research should focus on two main areas of ventilation. First, are providers consistently achieving ventilation targets during cardiac arrest management? Second, what is the relationship between ventilation parameters and patient outcomes? Optimization of ventilation targets and ensuring accurate delivery may lead to significantly improved outcomes from cardiac arrest.

Early studies examining real-time ventilation feedback mimic the introduction of real-time chest compression feedback devices. Although chest compression feedback devices have consistently shown improved performance in simulated and training environments, the results in clinical settings have been mixed. Most clinical studies continue to demonstrate improvements in chest compression quality metrics; however, they report inconsistent impacts on clinical outcomes. Interestingly, consistent across most studies, ventilation rate and chest compression rate appear to benefit most from real-time feedback, whereas chest compression depth and ventilation volume appear to be more challenging to improve.

Consistent with the introduction of chest-compression feedback, it is unlikely that implementation of ventilation devices without appropriate training and ongoing quality assurance/improvement programs will be sufficient to improve ventilation quality. Implementation of new technology requires time to adopt the technology and an ongoing cycle of evaluation and feedback. Feedback devices should be implemented as part of a larger program aimed at high-performance resuscitation. In addition, implementation of ventilation feedback involves additional challenges, as little is known about the optimal ventilation requirements for patients during cardiac arrest. Research must now determine whether improved technical compliance translates to better patient outcomes, including ROSC, survival to discharge, and neurologic recovery.

Airway Closure and the Use of Positive End Expiratory Pressure

Airway closure, in which there is interrupted communication between the proximal airway opening and distal alveoli, can result in a lack of insufflation of alveoli and a reduction in gas exchange despite manual or mechanical ventilation (MV) efforts.⁵⁹ The effect of airway closure has been described in other clinical conditions, such as obesity,⁶⁰ asthma,⁶¹ and ARDS, especially in situations with use of low positive end-expiratory pressure (PEEP).⁶² This same phenomenon has been described in

patients with cardiac arrest after intubation.^{59,63} Physiologically, it is hypothesized that chest compressions result in a reduction in lung volume below the end-expiratory volume, or functional residual capacity, which causes closure of the small distal airways.^{59,63} Airway closure may alter measurements of driving pressure, underestimate lung compliance, and cause alveolar collapse, resulting in lung damage and poor ventilation in patients with cardiac arrest.

Several studies have demonstrated that the presence of airway closure can be detected by capnogram waveform patterns during ET_{CO₂} monitoring.^{59,63–66} As discussed previously, chest compressions can impact ET_{CO₂} interpretation. In some instances, chest compressions cause oscillations in the capnogram waveforms (Fig. 2).^{63,66} This oscillating pattern is what would be expected in an airway without airway closure as air enters and exits the system during decompression and compression of the chest. In other instances, there are no oscillations in the capnogram waveform with chest compressions. This stable capnogram tracing represents airway closure. Nonoscillating patterns (airway closure) have been identified in up to 60% of patients in cardiac arrest.⁵⁹ These patterns in capnogram waveforms change over the course of resuscitation, with an increase in nonoscillating patterns (airway closure) occurring later in the resuscitation, suggesting that ventilation may become less effective with longer duration of CPR.

Furthermore, quantification of the oscillations in ET_{CO₂} waveforms, termed the airway opening index (AOI), has been described as a ratio of the average change in ET_{CO₂} owing to chest compressions compared with the maximum ET_{CO₂}. The AOI is associated with ROSC. Specifically, higher AOI is associated with increased rates of ROSC, presumably because open airways allow adequate oxygen delivery to and CO₂ removal from the alveoli.⁶⁷

One way to reduce the impact of airway closure is through the application of PEEP. The use of PEEP can increase alveolar recruitment and end-expiratory volume, preventing distal airway collapse and improving ventilation. The use of PEEP improves arterial blood gas measurements (pH, PO₂, PCO₂) in non-cardiac arrest populations at risk of alveolar collapse and in patients in cardiac arrest.

The cardiopulmonary effects of PEEP present a complex balance during low-flow states; thus, the current use of PEEP during CPR remains controversial. PEEP can lead to increased intrathoracic pressure and subsequent reduction in venous return. An animal study reported that PEEP levels exceeding 10 cm H₂O can reduce cardiac output by 15% to 25% during CPR.⁶⁸

However, completely avoiding PEEP can lead to alveolar derecruitment, which may increase pulmonary shunt fraction and worsen oxygen delivery.⁶⁹ A PEEP value

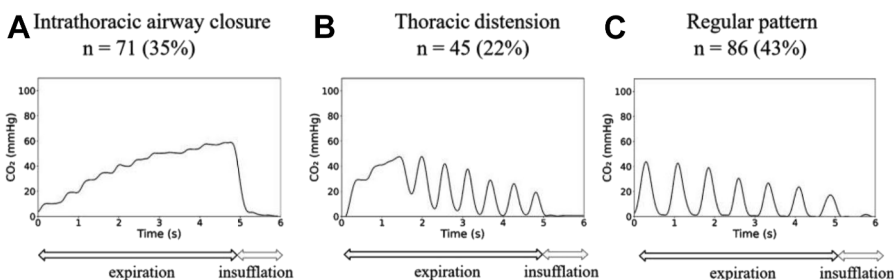


Fig. 2. ET_{CO₂} waveform patterns. (Lesimple, A., Fritz, C., Hutin, A. et al. A novel capnogram analysis to guide ventilation during cardiopulmonary resuscitation: clinical and experimental observations. Crit Care 26, 287 (2022). <https://doi.org/10.1186/s13054-022-04156-0>.⁶⁵)

greater than 5 cm H₂O is associated with improved oxygenation, although PEEP values between 0 and 5 cm H₂O have been associated with optimal oxygen delivery while maintaining cardiac output during CPR.⁶⁸

The potential benefits of PEEP during cardiac arrest resuscitation require investigation, as current recommendations suggest zero to low-pressure PEEP to avoid impaired venous return, but this may lead to airway closure, atelectasis, and impaired gas exchange. Similarly, research should determine optimal fraction of inspired oxygen (FiO₂) settings, as current recommendations suggest 100% oxygen, but the potential for oxidative injury with prolonged exposure to high oxygen concentrations must be balanced against the need for maximal oxygenation during low cardiac output states.

One novel area of evaluation is in using capnogram waveform patterns to guide the use of PEEP and Vt during continuous chest compressions. Lesimple and colleagues⁶⁵ identified three patterns of capnogram waveforms: (1) overdistension; (2) regular; (3) airway closure. In a mixed study of bench science and clinical observational research, they calculated a “distention ratio” that was able to separate ETco₂ waveform patterns into these three groups. Using animal models, they demonstrated that the distention ratio calculated from capnogram waveform patterns of thoracic distention were negatively correlated with mean arterial blood pressure and cerebral perfusion pressure, suggesting that higher distention ratios resulted in impaired circulation during cardiac arrest. The investigators postulated that capnogram patterns could aid in tailoring Vt and PEEP to balance between thoracic distention and airway closure for optimizing ventilation and hemodynamics. Nonoscillating patterns of airway closure indicate potential need to increase PEEP, and oscillating patterns of thoracic distention indicate potential need to decrease Vt.

Use of Mechanical Ventilation for Cardiac Arrest Resuscitation

MV, whether pressure control or volume control, allows providers more precise monitoring and control over the delivery of ventilations compared with manual ventilation. MV provides several advantages compared with manual ventilation. In task-saturated and resource-limited settings, common during CPR, MV can provide consistent ventilations while reducing the impact of human error and the need for additional providers. Chest compressions, however, can lead to an increase in peak inspiratory pressure, which can lead to increased risk of lung injury and make it challenging to deliver adequate Vt. Providers must be aware, if using MV during cardiac arrest, to adjust pressure limits and inactivate the inspiratory trigger to avoid the impact of chest compressions on interruptions in ventilation delivery (Table 1). There are limited data on the use of MV during CPR. A small, single-center RCT (n = 60) of patients with non-traumatic cardiac arrest in the emergency department found no difference in arterial blood oxygenation between MV and manual ventilation (36.5 mm Hg vs 29.0 mm Hg, respectively). They also found no difference in rates of ROSC or survival between groups.⁷⁰ A second pilot RCT (n = 60) found that the use of MV was feasible during cardiac arrest resuscitation. The investigators found no difference in ROSC (56.7% in MV vs 43.3% in manual ventilation; *P* = .439) and no differences in blood-gas measurements (pH, PO₂, Pco₂, bicarbonate, lactate) between groups.⁷¹ There was a significant decrease in Vt and minute ventilation observed in the MV group.⁷¹ Although again showing promise, further research is needed to determine the utility and impact on patient outcomes of using MV during cardiac arrest resuscitation.

Manual Resuscitators

Today's manual resuscitators were designed more than 70 years ago, and little has changed. Several manufacturers have worked to address hyperventilation, volutrauma,

Table 1
Suggested settings for mechanical ventilation

Ventilator Mode	Pressure Control
Pressure control	15 cm H ₂ O
Positive end expiratory pressure	5 cm H ₂ O
Ventilation rate	10 per minute
Inspiratory time	1 s (I:E 1:5)
Inspiratory trigger	OFF
Pressure alarms	Maximum
Tidal volume	Off/>600 mL
Minute ventilation	< 1.0/>15 L/min
Fio ₂	100%

and barotrauma by challenging the original design. These design changes have targeted the known injuries associated with manual ventilation. To date, manufacturers have limited or allowed the setting of stroke volumes (limiting V_t) and/or restrict inflow or outflow of the bag to limit hyperventilation owing to high RR. To improve provider comfort, bag-mask devices have been created more ergonomically friendly for smaller hands or to use for longer durations. Many bag-mask devices can come with pressure manometers and pressure pop-off valves, but these often add to the cost and are not required to purchase.

Automated Resuscitators

COVID-19 renewed interest and innovation in the gas-powered resuscitator market when mechanical ventilators were predicted to be in short supply. Most provide automated pressure-cycled ventilation from a 50 psi gas source without the need for electricity. Although pressure-cycled ventilation helps control airway pressure and potentially reduces barotrauma, it does little to prevent volutrauma without monitoring V_ts and titrating pressures. Chest rise and fall can be leveraged as a visual of adequate ventilation, and the automation has the potential to allow for two-hand mask technique or other tasks if an advanced airway is placed. Improvements in these devices have revolved around ease of use, lower pressure adjustments, and reduction in oxygen consumption by the reduction of Fio₂ through a Venturi system.

FUTURE DIRECTION

Personalized Ventilation/Resuscitation

It is unlikely that a one-size-fits-all approach to resuscitation optimizes care for all patients. This is also true for ventilation. As evidence and technology continue to advance, opportunities to tailor ventilations based on patient-specific demographics and cardiac arrest characteristics may be possible. Ventilations for patients not in cardiac arrest are commonly delivered based on calculation of predicted body weight (PBW), which is determined by a patient's height. This may also provide an appropriate initial estimate for patients in cardiac arrest. Although blood gases may be challenging to use to guide resuscitation, other patient-level factors may help to individualize ventilations during cardiac arrest. Part of this may lie in better understanding of capnogram waveforms to indicate airway closure or in more

sophisticated technology to provide other metrics of ventilation quality. As with most other patient conditions, initial ventilation following ROSC is provided based on calculations of PBW and modified based on patient condition. A goal of normocapnia is currently recommended¹⁷; however, it is not clear if mild hypercapnia may also be appropriate for some patients. A large clinical trial found that targeting mild hypercapnia in the intensive care unit following out-of-hospital cardiac arrest (OHCA) did not result in better outcomes compared with normocapnia.⁷² However, other studies have found some potential benefits with mild hypercapnia. Melberg and colleagues⁷³ in a limited substudy demonstrated that in resuscitated adult patients with OHCA that mild hypercapnic patients had improved cardiac performance and mixed venous saturations without the negative consequences of elevated PVR and worsening right ventricular dysfunction. Ventilation targets can often be achieved with a little knowledge of V_t s, minute ventilation, and normal RRs while monitoring ET_{CO_2} .

Important Knowledge Gaps

Current evidence suggests that effective ventilation during cardiac arrest may significantly impact survival outcomes; yet critical knowledge gaps remain. Research on ventilation during CPR lags behind chest compression research, with limited high-quality data guiding current recommendations. The recently developed ventilation-measuring devices that can be used in the out-of-hospital setting should help answer some of these knowledge gaps.

Titrating ventilation parameters to physiologic endpoints, such as ET_{CO_2} , arterial blood gases, or cerebral oxygenation, may yield better outcomes than fixed parameter targets. Because cardiac output during CPR is only 25% to 33% of normal, the optimal minute ventilation for maintaining adequate gas exchange while minimizing adverse hemodynamic effects may differ significantly from conventional ventilation approaches.

Future research should investigate whether specific combinations of rate and volume are optimal for different cardiac arrest scenarios, patient characteristics, or phases of resuscitation. Research on ventilation parameters beyond rate and volume remains particularly sparse. Inspiratory time, inspiratory-to-expiratory ratio, airway pressures, and the interaction between chest compressions and ventilation may be associated with patient outcomes during cardiac arrest and should be explored.

Current ventilation feedback technologies primarily use differential pressure-based flow sensors placed between the ventilation bag and airway to measure and display RR and ventilation volume. Future research should compare different feedback modalities to determine which approaches provide the most usable guidance with minimal cognitive burden. Feedback devices may divide provider attention between patient and device, potentially increasing cognitive workload during resuscitation efforts. Investigation into alternative display methods, including heads-up displays, auditory feedback, or tactile feedback, could potentially address this limitation.

SUMMARY

Ventilation is an important, yet understudied, aspect of cardiac arrest resuscitation. Hyperventilation and hypoventilation can both be detrimental in cardiac resuscitation; however, what constitutes optimal ventilation is not known. Current technology is limited in guiding ventilation. Furthermore, appropriate targets to optimize ventilation in cardiac arrest are not well described. Advancements in technology have

created an opportunity to significantly improve the understanding of ventilation in cardiac arrest.

CLINICS CARE POINTS

- Ventilation is a critical component of cardiopulmonary resuscitation.
- The optimal ventilation strategy during resuscitation is not known.
- Providers are often not consistent with best practice recommendations for ventilation during resuscitation.

DISCLOSURE

Ian is Senior Science Editor for the American Heart Association and Chair of the Advanced Life Support Guidelines for the International Liaison Committee on Resuscitation. Ian has received funding from ZOLL for research related to ventilation during cardiac arrest. Ian has received speaker honorarium from ZOLL related to ventilation during cardiac arrest.

REFERENCES

1. Slutsky AS, Ranieri VM. Ventilator-induced lung injury. *N Engl J Med* 2013;369(22):2126–36.
2. Patil KD, Halperin HR, Becker LB. Cardiac arrest: resuscitation and reperfusion. *Circ Res* 2015;116(12):2041–9.
3. Ranieri VM, Suter PM, Tortorella C, et al. Effect of mechanical ventilation on inflammatory mediators in patients with acute respiratory distress syndrome: a randomized controlled trial. *JAMA* 1999;282(1):54–61.
4. Aufderheide TP, Lurie KG. Death by hyperventilation: a common and life-threatening problem during cardiopulmonary resuscitation. *Crit Care Med* 2004;32(9 Suppl):S345–51.
5. Aufderheide TP, Sigurdsson G, Pirralo RG, et al. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. *Circulation* 2004;109(16):1960–5.
6. Fortune JB, Feustel PJ, Graca L, et al. Effect of hyperventilation, mannitol, and ventriculostomy drainage on cerebral blood flow after head injury. *J Trauma* 1995;39(6):1091–7 [discussion: 1097–9].
7. Godoy DA, Seifi A, Garza D, et al. Hyperventilation therapy for control of posttraumatic intracranial hypertension. *Front Neurol* 2017;8:8250.
8. Curley G, Kavanagh BP, Laffey JG. Hypocapnia and the injured brain: more harm than benefit. *Crit Care Med* 2010;38(5):1348–59.
9. Vanwulpen M, Wolfskeil M, Duchatelet C, et al. Do manual chest compressions provide substantial ventilation during prehospital cardiopulmonary resuscitation? *Am J Emerg Med* 2021;39:39129–31.
10. McDannold R, Bobrow BJ, Chikani V, et al. Quantification of ventilation volumes produced by compressions during emergency department cardiopulmonary resuscitation. *Am J Emerg Med* 2018;36(9):1640–4.
11. Bobrow BJ, Ewy GA, Clark L, et al. Passive oxygen insufflation is superior to bag-valve-mask ventilation for witnessed ventricular fibrillation out-of-hospital cardiac arrest. *Ann Emerg Med* 2009;54(5):656–62.e1.

12. Lurie KG, Nemergut EC, Yannopoulos D, et al. The physiology of cardiopulmonary resuscitation. *Anesth Analg* 2016;122(3):767–83.
13. Prause G, Zoidl P, Eichinger M, et al. Mechanical ventilation with ten versus twenty breaths per minute during cardio-pulmonary resuscitation for out-of-hospital cardiac arrest: a randomised controlled trial. *Resuscitation* 2023;187:109765.
14. Sutton RM, Reeder RW, Landis WP, et al, Eunice Kennedy Shriver National Institute of Child Health and Human Development Collaborative Pediatric Critical Care Research Network CPCCRN. Ventilation rates and pediatric in-hospital cardiac arrest survival outcomes. *Crit Care Med* 2019;47(11):1627–36.
15. Gazmuri RJ, Ayoub IM, Radhakrishnan J, et al. Clinically plausible hyperventilation does not exert adverse hemodynamic effects during CPR but markedly reduces end-tidal PCO₂. *Resuscitation* 2012;83(2):259–64.
16. Lopez J, Fernandez SN, Gonzalez R, et al. Different respiratory rates during resuscitation in a pediatric animal model of asphyxial cardiac arrest. *PLoS One* 2016;11(9):e0162185.
17. Panchal AR, Bartos JA, Cabanas JG, et al, Adult Basic and Advanced Life Support Writing Group. Part 3: adult basic and advanced life support: 2020 American heart association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation* 2020;142(16_suppl_2):S366–468.
18. Olasveengen TM, Semeraro F, Ristagno G, et al. European resuscitation council guidelines 2021: basic life support. *Resuscitation* 2021;161:98–114.
19. Berg KM, Bray JE, Ng KC, et al, Collaborators. 2023 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations: summary from the basic life support; advanced life support; pediatric life support; neonatal life support; education, implementation, and teams; and first aid task forces. *Circulation* 2023;148(24):e187–280.
20. Greif R, Bray JE, Djarv T, et al. 2024 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations: summary from the basic life support; advanced life support; pediatric life support; neonatal life support; education, implementation, and teams; and first aid task forces. *Circulation* 2024;150(24):e580–687.
21. Nichol G, Leroux B, Wang H, et al, ROC Investigators. Trial of continuous or interrupted chest compressions during CPR. *N Engl J Med* 2015;373(23):2203–14.
22. McInnes AD, Sutton RM, Orioles A, et al. The first quantitative report of ventilation rate during in-hospital resuscitation of older children and adolescents. *Resuscitation* 2011;82(8):1025–9.
23. Guidelines for cardiogeny cardiac care committee and subcommitpulmonary resuscitation and emergency cardiac care. Emertees, American heart association. Part I. Introduction. *JAMA* 1992;268(16):2171–83.
24. Dorges V, Ocker H, Hagelberg S, et al. Smaller tidal volumes with room-air are not sufficient to ensure adequate oxygenation during bag-valve-mask ventilation. *Resuscitation* 2000;44(1):37–41.
25. Drennan IR, McLeod SL, Cheskes S. Randomized controlled trials in resuscitation. *Resusc Plus* 2024;18:100582.
26. Idris AH, Aramendi Ecenarro E, Leroux B, et al. Bag-valve-mask ventilation and survival from out-of-hospital cardiac arrest: a multicenter study. *Circulation* 2023;148(23):1847–56.
27. Davidovic L, LaCovey D, Pitetti RD. Comparison of 1- versus 2-person bag-valve-mask techniques for manikin ventilation of infants and children. *Ann Emerg Med* 2005;46(1):37–42.

28. Hart D, Reardon R, Ward C, et al. Face mask ventilation: a comparison of three techniques. *J Emerg Med* 2013;44(5):1028–33.
29. Otten D, Liao MM, Wolken R, et al. Comparison of bag-valve-mask hand-sealing techniques in a simulated model. *Ann Emerg Med* 2014;63(1):6–12.e3.
30. Gerstein NS, Carey MC, Braude DA, et al. Efficacy of facemask ventilation techniques in novice providers. *J Clin Anesth* 2013;25(3):193–7.
31. Snyder BD, Van Dyke MR, Walker RG, et al. Association of small adult ventilation bags with return of spontaneous circulation in out of hospital cardiac arrest. *Resuscitation* 2023;193:109991.
32. Baskett P, Nolan J, Parr M. Tidal volumes which are perceived to be adequate for resuscitation. *Resuscitation* 1996;31(3):231–4.
33. Bengner JR, Kirby K, Black S, et al. Effect of a strategy of a supraglottic airway device vs tracheal intubation during out-of-hospital cardiac arrest on functional outcome: the AIRWAYS-2 randomized clinical trial. *JAMA* 2018;320(8):779–91.
34. Jabre P, Penalzoza A, Pinero D, et al. Effect of bag-mask ventilation vs endotracheal intubation during cardiopulmonary resuscitation on neurological outcome after out-of-hospital cardiorespiratory arrest: a randomized clinical trial. *JAMA* 2018;319(8):779–87.
35. Wang HE, Schmicker RH, Daya MR, et al. Effect of a strategy of initial laryngeal tube insertion vs endotracheal intubation on 72-hour survival in adults with out-of-hospital cardiac arrest: a randomized clinical trial. *JAMA* 2018;320(8):769–78.
36. Gutierrez JJ, Sandoval CL, Leturiondo M, et al. Contribution of chest compressions to end-tidal carbon dioxide levels generated during out-of-hospital cardiopulmonary resuscitation. *Resuscitation* 2022;179:225–32.
37. Chandran K, Algaze Gonzalez IM, Wang S, et al. Chest decompressions - the driver of CPR efficacy: exploring the relationship between compression rate, depth, recoil velocity, and end-tidal CO₂. *Prehosp Emerg Care* 2025;29(2):154–61.
38. Einav S, Bromiker R, Weiniger CF, et al. Mathematical modeling for prediction of survival from resuscitation based on computerized continuous capnography: proof of concept. *Acad Emerg Med* 2011;18(5):468–75.
39. Kodali BS, Urman RD. Capnography during cardiopulmonary resuscitation: current evidence and future directions. *J Emerg Trauma Shock* 2014;7(4):332–40.
40. Levine RL, Wayne MA, Miller CC. End-tidal carbon dioxide and outcome of out-of-hospital cardiac arrest. *N Engl J Med* 1997;337(5):301–6.
41. Wang AY, Huang CH, Chang WT, et al. Initial end-tidal CO₂ partial pressure predicts outcomes of in-hospital cardiac arrest. *Am J Emerg Med* 2016;34(12):2367–71.
42. Nassal MMJ, Elola A, Aramendi E, et al. Temporal trends in end-tidal capnography and outcomes in out-of-hospital cardiac arrest: a secondary analysis of a randomized clinical trial. *JAMA Netw Open* 2024;7(7):e2419274.
43. Paiva EF, Paxton JH, O'Neil BJ. The use of end-tidal carbon dioxide (ETCO₂) measurement to guide management of cardiac arrest: a systematic review. *Resuscitation* 2018;12:31–7.
44. Morgan RW, Reeder RW, Bender D, et al. ICU-RESUS and Eunice Kennedy Shriver National Institute of Child Health and Human Development Collaborative Pediatric Critical Care Research Network CPCCRN Investigator Groups. Associations between end-tidal carbon dioxide during pediatric cardiopulmonary resuscitation, cardiopulmonary resuscitation quality, and survival. *Circulation* 2024;149(5):367–78.

45. Ruiz de Gauna S, Gutierrez JJ, Ruiz J, et al. The impact of ventilation rate on end-tidal carbon dioxide level during manual cardiopulmonary resuscitation. *Resuscitation* 2020;156:215–22.
46. Callahan M, Barton C, Matthey M. Effect of epinephrine on the ability of end-tidal carbon dioxide readings to predict initial resuscitation from cardiac arrest. *Crit Care Med* 1992;20(3):337–43.
47. Martin GB, Gentile NT, Paradis NA, et al. Effect of epinephrine on end-tidal carbon dioxide monitoring during CPR. *Ann Emerg Med* 1990;19(4):396–8.
48. Roh YI, Kim HI, Kim SJ, et al. End-tidal carbon dioxide after sodium bicarbonate infusion during mechanical ventilation or ongoing cardiopulmonary resuscitation. *Am J Emerg Med* 2024;76:211–6.
49. Grmec S, Lah K, Tusek-Bunc K. Difference in end-tidal CO₂ between asphyxia cardiac arrest and ventricular fibrillation/pulseless ventricular tachycardia cardiac arrest in the prehospital setting. *Crit Care* 2003;7(6):R139–44.
50. Gould JR, Campana L, Rabickow D, et al. Manual ventilation quality is improved with a real-time visual feedback system during simulated resuscitation. *Int J Emerg Med* 2020;13(1):18.
51. Charlton K, McClelland G, Millican K, et al. The impact of introducing real time feedback on ventilation rate and tidal volume by ambulance Clinicians in the north east in cardiac arrest simulations. *Resusc Plus* 2021;6:100130.
52. D'Agostino F, Agro FE, Petrosino P, et al. Are instructors correctly gauging ventilation competence acquired by course attendees? *Resuscitation* 2024;200:110240.
53. Heo S, Yoon SY, Kim J, et al. Effectiveness of a real-time ventilation feedback device for guiding adequate minute ventilation: a manikin simulation study. *Medicina (Kaunas)* 2020;56(6):278.
54. Kim JW, Park SO, Lee KR, et al. Efficacy of Amflow(R), a real-time-portable feedback device for delivering appropriate ventilation in critically ill patients: a randomised, controlled, cross-over simulation study. *Emerg Med Int* 2020;2020:5296519.
55. Lyngby RM, Clark L, Kjoelbye JS, et al. Higher resuscitation guideline adherence in paramedics with use of real-time ventilation feedback during simulated out-of-hospital cardiac arrest: a randomised controlled trial. *Resusc Plus* 2021;5:100082.
56. Scott JB, Schneider JM, Schneider K, et al. An evaluation of manual tidal volume and respiratory rate delivery during simulated resuscitation. *Am J Emerg Med* 2021;45:446–50.
57. Debaty G, Johnson NJ, Dewan M, et al. Real-time ventilation quality feedback devices efficacy in out-of-hospital cardiac arrest: a scoping review. *Resuscitation Plus* 2025;101069.
58. Drennan IR, Lee M, Heroux JP, et al. The impact of real-time feedback on ventilation quality during out-of-hospital cardiac arrest: a before-and-after study. *Resuscitation* 2024;204:110381.
59. Charbonney E, Grieco DL, Cordioli RL, et al, CAVIAR Group. Ventilation during cardiopulmonary resuscitation: what have we learned from models? *Respir Care* 2019;64(9):1132–8.
60. Coudroy R, Vimpere D, Aissaoui N, et al. Prevalence of complete airway closure according to body mass index in acute respiratory distress syndrome. *Anesthesiology* 2020;133(4):867–78.
61. King GG, Eberl S, Salome CM, et al. Differences in airway closure between normal and asthmatic subjects measured with single-photon emission computed tomography and technegas. *Am J Respir Crit Care Med* 1998;158(6):1900–6.

62. Chen L, Del Sorbo L, Grieco DL, et al. Airway closure in acute respiratory distress syndrome: an underestimated and misinterpreted phenomenon. *Am J Respir Crit Care Med* 2018;197(1):132–6.
63. Cordioli RL, Lyazidi A, Rey N, et al. Impact of ventilation strategies during chest compression. An experimental study with clinical observations. *J Appl Physiol* 1985 2016;120(2):196–203.
64. Grieco DL, L JB, Drouet A, et al. Intrathoracic airway closure impacts CO(2) signal and delivered ventilation during cardiopulmonary resuscitation. *Am J Respir Crit Care Med* 2019;199(6):728–37.
65. Lesimple A, Fritz C, Hutin A, et al, CAVIAR Cardiac Arrest, Ventilation International Association for Research Group. A novel capnogram analysis to guide ventilation during cardiopulmonary resuscitation: clinical and experimental observations. *Crit Care* 2022;26(1):287.
66. Leturiondo M, Ruiz de Gauna S, Ruiz JM, et al. Influence of chest compression artefact on capnogram-based ventilation detection during out-of-hospital cardiopulmonary resuscitation. *Resuscitation* 2018;124:63–8.
67. Bhandari S, Coult J, Counts CR, et al. Investigating the airway opening index during cardiopulmonary resuscitation. *Resuscitation* 2022;178:96–101.
68. Levenbrow Y, Hossain MJ, Keith JP, et al. The effect of positive end-expiratory pressure on cardiac output and oxygen delivery during cardiopulmonary resuscitation. *Intensive Care Med Exp* 2020;8(1):36.
69. Renz M, Mullejans L, Riedel J, et al. High PEEP levels during CPR improve ventilation without deleterious haemodynamic effects in pigs. *J Clin Med* 2022;11(16).
70. Tangpaisarn T, Tosibphanom J, Sata R, et al. The effects of mechanical versus bag-valve ventilation on gas exchange during cardiopulmonary resuscitation in emergency department patients: a randomized controlled trial (CPR-VENT). *Resuscitation* 2023;193:109966.
71. Shin J, Lee HJ, Jin KN, et al. Automatic mechanical ventilation vs manual bag ventilation during CPR: a pilot randomized controlled trial. *Chest* 2024;166(2): 311–20.
72. Eastwood G, Nichol AD, Hodgson C, et al, TAME Study Investigators. Mild hypercapnia or normocapnia after out-of-hospital cardiac arrest. *N Engl J Med* 2023; 389(1):45–57.
73. Melberg MB, Flaa A, Andersen GO, et al. Cardiovascular changes induced by targeted mild hypercapnia after out of hospital cardiac arrest. A sub-study of the TAME cardiac arrest trial. *Resuscitation* 2023;193:109970.